Spontaneous splenic rupture with massive bleeding during Pringle maneuver in laparoscopic liver resection

Rotura esplénica espontánea con hemorragia masiva durante la maniobra de Pringle en cirugía hepática laparoscópica

The Pringle maneuver, described by JH Pringle in 1908 for liver resection surgery, consists of total occlusion of the hepatic portal pedicle at the level of the hepatoduodenal ligament. Clamping can be performed continuously or intermittently with periods of ischemia of 15–20 min alternated with 5 min of reperfusion, and with a variable duration depending on the quality of the organ.[1] It is a safe and effective technique for the control of intraoperative bleeding as well as for the preservation of liver function during the immediate postoperative period.[2] In addition, a significant decrease in adverse events has been observed in patients to whom it is applied.[3] However, isolated cases of spontaneous splenic rupture have been described secondary to performing this maneuver, and early diagnosis is essential to control bleeding.[4–7] Because of the fatal consequences of this exceptional phenomenon, all surgeons, in particular liver surgeons, should be aware of this entity when intraoperative bleeding is detected after a Pringle maneuver in the absence of an obvious hepatic origin.

We present the case of a 49-year-old male patient with a history of thoracic-lumbar melanoma treated surgically. During follow-up, a single 15-mm hepatic metastasis was diagnosed in the dome of segment VII as well as right axillary lymphadenopathies, the largest measuring 6 cm, compatible with lymph node metastasis. No vascular abnormalities or collateral circulation were identified on the preoperative nuclear magnetic resonance. After evaluation by the Multidisciplinary Committee, joint elective surgical intervention was decided (laparoscopic hepatic segmentectomy and lymphadenectomy in the same operation). The patient was placed in the modified Lloyd-Davies position, with the right arm abducted and flexed over the head with the help of a support, elevation of the right hemibody to around 30° with a cushion under the dorsal spine, and inverted Trendelenburg. Four trocars were inserted under direct vision, and insufflation of the pneumoperitoneum was performed without incident. At the end of the segment VII segmentectomy and after applying the extracorporeal Pringle maneuver in three consecutive intervals (a total of 45 min of clamping with 5 min of reperfusion between each interval), the patient presented an episode of hemodynamic instability with sustained hypotension. After ruling out the possibility of an air embolism as an etiological agent and observing a drop of 6 hemoglobin points with no active bleeding detected in the surgical bed, we decided to convert to open surgery using a j-shaped Makuuchi, finding abundant clots around the spleen, suggestive of spontaneous subcapsular rupture. Urgent splenectomy was performed, and the patient required polytransfusion (5 units packed red blood cell, 2 units fresh frozen plasma, and one unit of packed platelets plus antifibrinolytic agents) to restore hemodynamic stability. The pathological analysis described the surgical piece as splenic decapsulation in the context of subcapsular hemorrhage and the presence of foci of accompanying inflammatory necrosis (Fig. 1). After a correct immediate postoperative period, right axillary lymphadenectomy was performed in a second operation. After satisfactory evolution, the patient was discharged to home on the 10th day of hospitalization, with no postoperative incidents.

The etiological factors[8] of spontaneous splenic rupture include increased pressure in the portal venous system.[4] Portal hypertension (PHT) is a syndrome characterized by a pathological increase in portal pressure, the most common cause of which is cirrhosis of the liver. Under physiological conditions, portal pressure is between 5 and 10 mmHg, with a portacaval gradient of less than 5 mmHg. Clinically significant PHT is defined as an increase in portal pressure above 10 mmHg or a portacaval gradient greater than 5 mmHg, a situation leading to possible complications.[4] The increase in vascular resistance results in the appearance of the aforementioned PHT which, after a brief initial period of hypodynamic splanchnic circulation, transforms the splanchnic bed into a hyperdynamic state. As a result, vasodilation and angiogenesis occur with the appearance of a portosystemic collateral circulation that is expressed as esophageal, gastric, paraumbilical, pararectal, retroperitoneal varices and splenorenal shunts[9] (Fig. 2).[4]

Hemodynamically, the Pringle maneuver implies increased cardiac output with elevated blood pressure and systemic vascular resistance. In addition, there is an increase in pressure at the portal level and through its collaterals, which under physiological conditions are collapsed. Patients with PHT are usually capable of compensating for the increase in pressure with a redistribution of splanchnic flow while, in the absence of this, a late adaptation occurs with an accumulation of blood at this level that leads to the appearance of the aforementioned phenomenon,[9] as happened in the case described.

In patients with carcinoma of colorectal origin and synchronous liver metastases, simultaneous resection of the primary tumor and liver lesions is technically feasible, with similar perioperative and oncological results.[10] Currently, there is controversy regarding the order of resection of lesions in combined surgery. During hepatectomy, the Pringle maneuver causes an increase in congestion of the splanchnic territory and consequently in mesenteric venous pressure, which is related to a higher probability of hemorr-

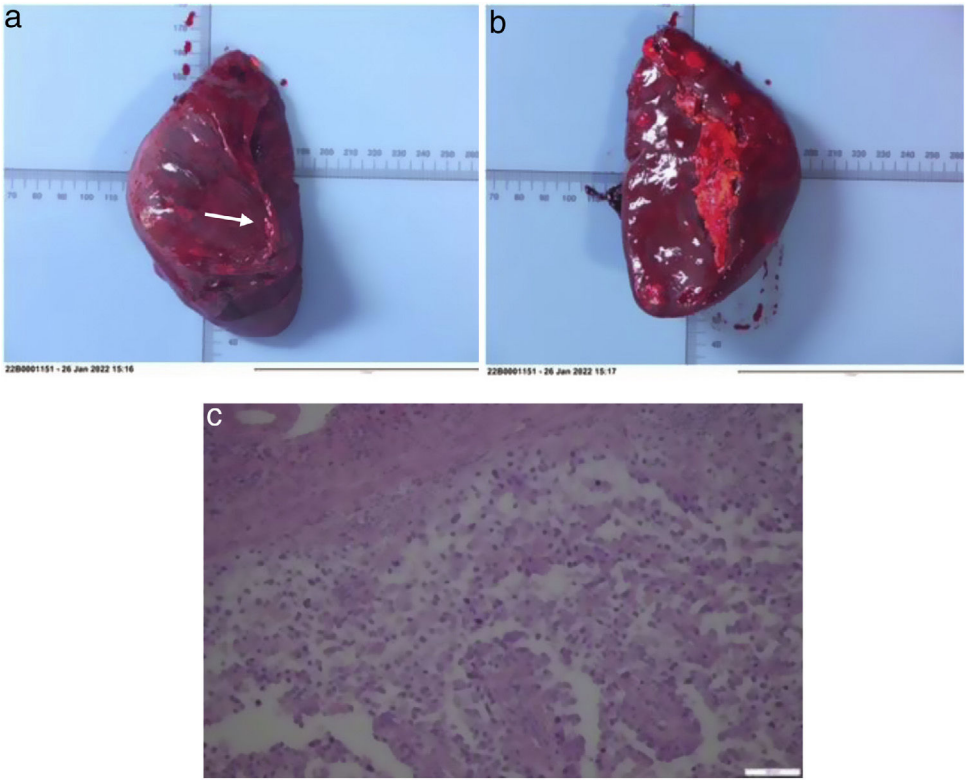

Figure 1 – a–b. Surgical piece: decapsulated spleen. The arrow (1a) points the detached splenic capsule. Figure 1-c. Splenic subcapsular hemorrhage and inflammatory necrosis areas.

hage during transection of the mesocolon.[10] In addition, significant intestinal congestion could lead to an anastomotic leak.[12] This situation could be avoided with restrictive blood volume control during hepatectomy.[10]

Published studies show that the Pringle maneuver is a safe and effective technique for the prevention of ischemia-reperfusion injury in liver resection surgery.[1,3] However, in the case of intraoperative hypovolemic shock without obvious hepatic bleeding, it is imperative to consider the possibility of spontaneous splenic rupture among the differential diagnoses. In this regard, the laparoscopic approach and the position of the patient in left lateral decubitus pose a challenge for surgeons since they can conceal the suspected diagnosis.

Case reports about this entity are rare, and only a single case has been described in laparoscopic liver surgery.[3] According to the literature consulted, the therapeutic management of these patients included, above all, rapid unclamping of the portal pedicle (essential to prevent the progression of splenic hemorrhage as well as possible omental injuries related to inadvertent adhesions, whose adhesiolysis could cause hemoperitoneum) in addition to adequate hemodynamic resuscitation with intensive fluid therapy.[3] Once the splenic origin of the hemorrhage was located, urgent splenectomy and massive polytransfusion were performed in most cases given the difficult hemostatic control due to splenic decapsulation (Table 1). We should mention the publication of a single case of spontaneous splenic rupture with hemodynamic stability maintained in laparoscopic liver surgery without the need for conversion or splenectomy due to rapid localization of the hemorrhage and effective control with blood products and hemostatic agents (Table 1).

In general terms, as long as the patient is hemodynamically stable and in the event of rapid localization of the splenic origin of the hemorrhage, control of the hemorrhage using hemostatic agents and hemoderivatives could be an option. Furthermore, maintaining high intra-abdominal pressure by adjusting the flow insufflated for the pneumoperitoneum could be effective in laparoscopic surgery.[3]

In the event of failure of the previous measures, bleeding of unknown origin or difficult-to-control bleeding, therapeutic management would include placing the patient in a supine position at 0° for rapid conversion to open surgery and a thorough examination of all abdominal quadrants. Regarding the choice of incision in the conversion to open surgery, the most appropriate options would provide rapid access to the abdominal cavity with wide exposure of the surgical field, such as supra-infraumbilical midline laparotomy, bilateral subcostal incision, or the j-shaped Makuuchi incision.[11] In our case, the choice of this latter incision was justified by the high suspicion of hemorrhage in the supramesocolic compartment. Also, since this incision is usually used in our hospital for open liver surgery, we decided to perform it because the liver transection had not yet been completed.

The decision for splenectomy must be made on an individual basis if effective control of bleeding is not achieved and the patient presents sustained hemodynamic

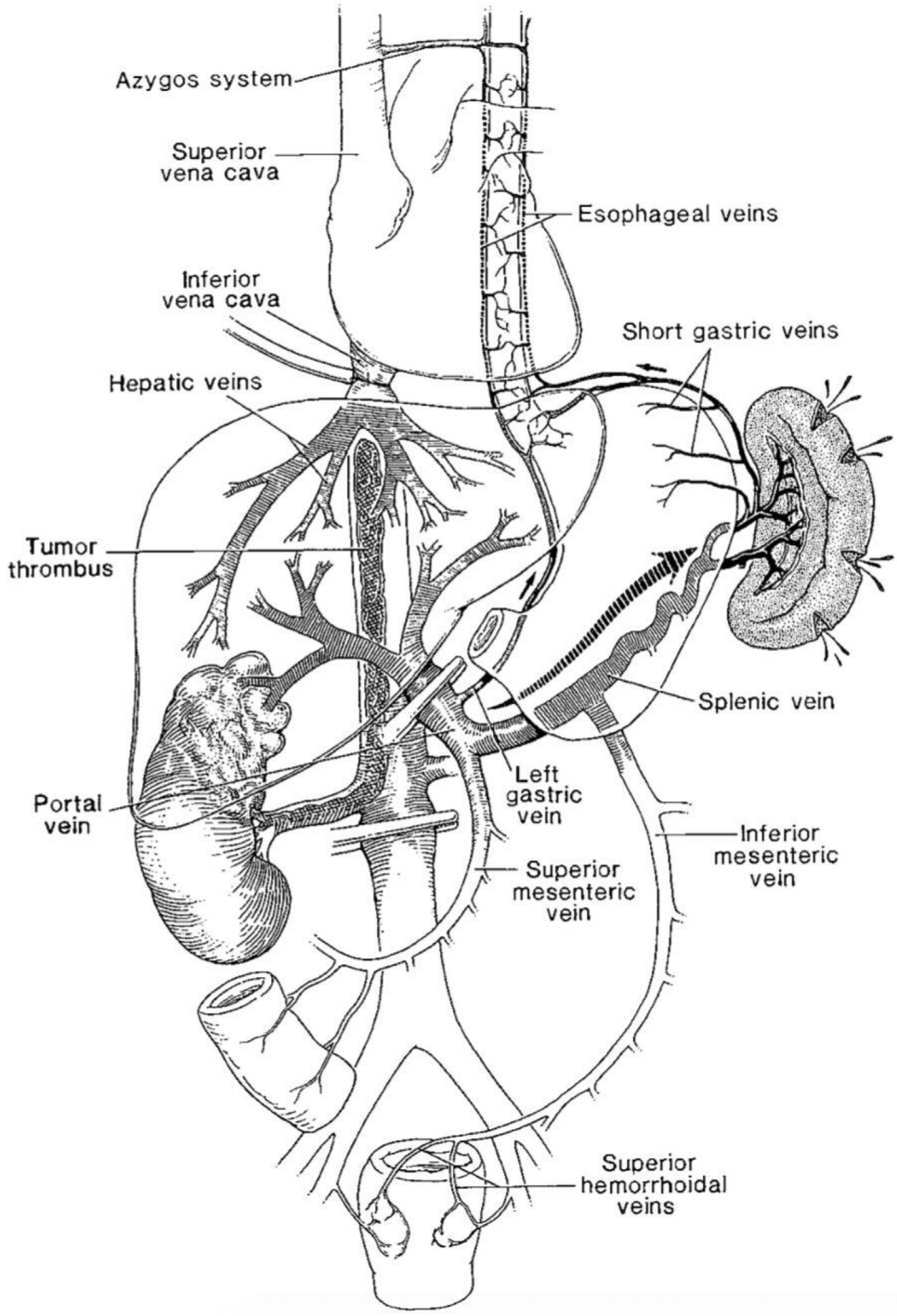

Figure 2 – Physiopathology of portal hypertension.[6]

instability.[3,12] Once the acute condition is resolved by splenectomy, it would be possible to complete the liver transection by performing the Pringle maneuver again (Table 1). If splenectomy was not performed and hemorrhage control with packing or hemostatic agents has been chosen, close monitoring is necessary during the clamping and unclamping periods to verify the absence of a new splenic hemorrhage.[3]

The diagnostic suspicion of this entity together with thorough exploration of the abdominal cavity by quadrants and rapid unclamping of the portal pedicle are fundamental measures in the initial approach. An expert surgeon could use laparoscopic management if the stability of the patient allows. However, in cases of hemodynamic instability, rapid conversion to open surgery with urgent splenectomy is mandatory.

Appendix A. Supplementary data

Supplementary material related to this article can be found, in the online version, at doi:https://doi.org/10.1016/j.ciresp.2022.10.021.

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http://dx.doi.org/10.1016//j.ciresp.2022.12.004
2173-5077/

High-grade dysplasia in the cystic duct after cholecystectomy

Displasia de alto grado en el conducto cístico tras colecistectomía

Laparoscopic cholecystectomy is one of the most frequently performed surgeries. High-grade dysplasia (HGD) in the cystic duct resection margin is uncommon after cholecystectomy for cholelithiasis (<0.1%) or acute cholecystitis (1%), and it is considered a precursor of gallbladder and bile duct cancer.[1,2] Therefore, when there is a finding of dysplasia, it is necessary to rule out concomitant tumor pathology, mainly multifocal biliary tumors (biliary intraductal papillary neoplasm, gallbladder adenocarcinoma and cholangiocarcinoma).[2] However, when faced with an isolated HGD with no clear signs of invasion, there is no therapeutic consensus.[3]